

residues 163 to 201 of SEQ ID NO:2, or to the region between said fourth cysteine rich domain of the p75 TNF receptor and the cell membrane, which region consists of the sequence of amino acid residues 201-257 of SEQ ID NO:2, with the proviso that said antigen binding portion is not that of a monoclonal antibody from the clone 67 (CNCM No. I-1368).

B' cond [Delete claim 16 without prejudice toward the continuation of prosecution in a continuing application.

REMARKS

Claims 13-15 presently appear in this case. No claims have been allowed. The official action of March 15, 2002, has now been carefully studied. Reconsideration and allowance are hereby respectfully urged.

Briefly, the present invention relates to a method for inhibiting the cytotoxic effect of TNF without blocking TNF binding to the p75 TNF receptor. This is accomplished by bringing to the vicinity of TNF a molecule which includes the antigen binding portion of antibodies which are specific to certain regions of the p75 TNF receptor, other than clone 67.

Claims 13-16 have been rejected under 35 U.S.C. §102(b) as being anticipated by the Wallach European patent 398 327. The examiner states that Wallach teaches the use of TNF receptor-specific antibodies, including the 67-specificity of claim 16 for various procedures, including the treatment of